4:15

866-2 Natural History of Donor-transmitted Atherosclerosis in Transplant Patients: Serial Intravascular Ultrasound Study

S.R. Kapadia, T.D. Crowe, K.M. Ziada, J.B. Young, R.E. Hobbs, V. Guetta, W.A. Magyar, S.E. Nissen, E.M. Tuzcu. *The Cleveland Clinic Foundation, Cleveland, Ohio, USA*

Background: The fate of donor-transmitted atherosclerotic coronary lesions in cardiac transplant recipients remains completely unkown.

Methods: Serial intravascular ultrasound was performed at baseline (4 weeks), 1 year and 2 years after transplantation (mean 28 ± 17, 370 ± 31, and 761 ± 49 days, respectively). For each examined site, maximal intimal thickness (Pmax) and intimal area were measured. Intimal area was defined as the difference between external elastic membrane and luminal cross-sectional areas. Donor-transmitted disease was defined as Pmax 0.5 mm. Lesions were classified as focal, when they involved less than the entire length of a segment as defined by the Coronary artery Surgery study (CASS).

Results: We examined 289 sites in 50 patients. At 29 sites in 22 patients (44%), at least one donor-transmitted atherosclerotic lesion was present by ultrasound. Donor-transmitted disease was mainly focal (26/29 or 90%). The Pmax and intimal area of baseline (4 week) lesions averaged 0.78 ± 0.26 mm and 3.02 ± 1.91 mm^2, respectively. At one year examination, there was a significant increase in Pmax and intimal area at previously identified sites with lesions now averaging 0.95 ± 0.45 mm and 6.46 ± 4.03 mm^2, $p < 0.02$ and <0.01, respectively. Major progression, defined as a Pmax increase 0.3 mm occurred at 7 sites (30%) in 6 patients. These lesions exhibited reduced locality by the one year exam (55% focal). Interestingly, at two year follow-up, the Pmax and intimal area in donor-transmitted lesions did not increase further, averaging 0.95 ± 0.45 mm and 6.14 ± 3.99 mm^2. Focality was largely unchanged compared to one year exam (62% focal).

Conclusion: Progression of donor atherosclerosis is significant during the first year after transplantation with minimal changes in the subsequent year. The apparent "burn out" of donor-transmitted disease between 1 and 2 years following transplantation suggests a relatively benign course for this phenomenon.

4:30

866-3 Morphologic Features of Vulnerable Coronary Atherosclerotic Plaque: Intravascular Ultrasound Demonstration With Prospective Follow-up Study

M. Yamagishi, M. Kijima, K. Ito, S. Nakatani, Y. Yasumura, K. Nakamura, S. Daikoku, K. Miyatake. *National Cardiovascular Center, Suita Osaka, Japan*

Background: Although evaluation of vulnerable atherosclerotic plaque is important, few data exist regarding the possible morphologic features of the plaque in clinical setting. Therefore, we performed the prospective follow-up study for the coronary atherosclerotic plaque that had been evaluated by intravascular ultrasound (IVUS).

Methods: From 105 patients, total 113 coronary sites where IVUS (30 MHz, 1,800 rpm) demonstrated *de novo* atherosclerotic lesions in the absence of angiographically significant stenosis were enrolled the follow-up protocol. The lesions consisted of 27 circumferential and 86 non-circumferential disease. Percent disease area that was determined by dividing the disease area by the total vessel area was 58 ± 13%. The lipid-rich core or intraplaque hemorrhage was defined as the presence of focal low echogenic area within the plaque, and the calcification as the high echogenic image with shadowing.

Results: During follow-up period of 18 ± 14 months (range 1 to 48), 12 patients had unstable angina or acute myocardial infarction 6.9 ± 10.4 months after IVUS examination. All the plaque related to the acute events consisted of non-circumferential lesions and 10 of 12 plaque contained the low echogenic areas within the disease images. The %plaque area of the occluded segments was greater (66 ± 10%) than that of non-occluded (57 ± 13%, $p < 0.05$) segments, although there was no statistical difference in lumen area between 2 groups (6.8 ± 2.9 vs 7.6 ± 3.8 mm^2). Four of the occluded plaque exhibited the augmented plaque motion during cardiac cycles in the IVUS images.

Conclusion: These results indicate that the larger non-circumferential plaque (>60%) with the intraplaque low echogenic areas could be vulnerable during the course of disease even though the lumen area is preserved. Whether the augmented plaque motion by IVUS can be related to the process of the plaque rupture should further be sought.

4:45

866-4 Intracoronary Ultrasound Guided Clinical Decision Making in Indeterminate Left Main Disease: 18 Month Follow-up Study

S.T. Higano, T.C. Yeo, A. Lerman, R.A. Nishimura, D.R. Holmes, Jr., *Mayo Clinic, Rochester, Minnesota, USA*

Left main coronary artery disease (LMCAD) is best treated with early surgical revascularization. LMCAD can be difficult to image with angiography due to the bifurcation of the left anterior descending and circumflex arteries, vessel foreshortening, contrast streaming, diffuse disease, and the ostial nature of LMCAD. Intracoronary ultrasound (ICUS) was used to evaluate angiographically indeterminate LMCAD in 48 pts. ICUS imaging was successful in 94% with three imaging failures occurring due to inability to cross the stenosis with first generation 5 Fr. catheters. There were no complications other than transient ST depression seen in 1 pt that was unsuccessfully imaged. Minimal or no LMCAD was found in 33 pts (Group 1). The remaining 12 pts (Group 2) had significant LMCAD and were referred for bypass surgery. Mean follow-up was 18 months.

	n	Area stenosis	Lumen area, mm^2	18 mos Cardiac mortality
Group 1	33	37 ± 14%	12 ± 4.9	3%
Group 2	12	68 ± 10%*	7.0 ± 2.1*	7%

* $p < 0.001$ vs Group 1

Lesions distal to the indeterminate LMCAD were dilated in 13 pts in Group 1 following the ICUS definition of the left main.

Conclusions: 1) Careful ICUS evaluation of indeterminate LMCAD is feasible and safe in selected pts, 2) ICUS can be used to define a low risk group with indeterminate LMCAD disease that has an excellent intermediate term outcome.

5:00

866-5 Serial Intravascular Ultrasound Evaluation of Remodeling in Transplant CAD: Influence of Disease Progression

K.M. Ziada, S.R. Kapadia, T.D. Crowe, G. Rincon, M. Goormastic, V. Guetta, S.E. Nissen, E.M. Tuzcu. *Cleveland Clinic Foundation, Cleveland, Ohio, USA*

Background: Serial ultrasound examination of transplant coronaries permits detailed study of arterial remodeling. However, measurement variability and changes in tone or flow may confound conclusions.

Methods: Ultrasound was performed within 154 coronary segments (32 patients, 55 arteries) within 4 weeks following transplantation and repeated at 1, 2, and 3 years. Measurements included lumen area, external elastic membrane (EEM) area, and maximum plaque thickness (Pmax). Segments were considered diseased if Pmax >0.5 mm. Control segments had Pmax <0.3 mm. Progression was defined as Pmax increase >0.3 mm. For each segment, a linear regression line was fit for lumen and EEM area over time. To account for variability, the average slope of the regression line was compared for diseased and control groups, and to a zero slope representing no change over time.

Results: In the diseased segments (n = 78), EEM area increased and lumen area decreased over time. The average slope was significantly different from control (EEM slope 0.78 ± 1.65 vs 0.38 ± 1.65, p = 0.05; lumen area slope −0.62 ± 1.38 vs 0.24 ± 1.53, p = 0.0009). The slopes of EEM and lumen areas in the controls were not significantly different from zero (p = 0.1 and 0.39 respectively). When the diseased segments were divided into progressive (n = 66) and non-progressive (n = 12) subgroups, the EEM area increase was larger in progressive subgroup.

Variable	Subgroup	Mean slope ± SD	p value vs. zero slope	p value vs. subgroups
EEM	progressive	1.05 ± 1.41	<0.001	0.002
Area	non-progressive	−0.68 ± 2.11	0.20	
Lumen	progressive	−0.65 ± 1.35	<0.001	0.64
Area	non progressive	−0.49 ± 1.62	0.15	

Conclusions: The EEM area of diseased segments shows significant increase over time which is most evident with disease progression. This demonstrates remodeling in very early transplant CAD.